

Correspondence

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advocated by Aldridge and Connors⁶ and Lauwerys and Louis-Ferdinand.⁷

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Bright red blood of Bhopal victims?

SIR,—In his letter (1984;43:502-4) Salmon elegantly discusses and refutes the theory of cyanide poisoning in Bhopal victims. Some limited animal work further denies that cyanide intoxication is involved in the intoxication with pure methyl isocyanate (MIC).¹ On the basis of the little information available, however, I am not even sure that the colour of the blood of the victims was really bright red. An early report about this issue is that of Ivan Fera "The day after" published in *The Illustrated Weekly of India* on 30 December 1984 and reprinted in a compilation of documents from Indian publications.² This article states: "By that afternoon (Dec 3rd 1984), the first autopsies had been performed. In many cases, the blood was purple red; the lungs ash coloured, and severely edematous" (p 49, my underlining). Only in the next paragraph is it said that "Heereshchandra, the head of the forensic department, felt that the evidence pointed strongly to cyanide. This was apparent in the bright red, almost pink colour of the blood and the unpleasant odour given off when the lungs were cut open—the oil of bitter almonds, the smell of cyanide" (p 50). Later sources, still quoting the same person, mention "cherry-red blood"³ or "dark, cherry-red blood," with lungs and other organs also red.⁴

It may seem futile to argue over different shades of red as reported by journalists, but the contradictory nature of the information in itself does little to support the whole cyanide issue. If the first quotation (purple blood) had been emphasised then the problem which had serious therapeutic and even sociopolitical implications^{4,5} could have been at least partly resolved.

The origin of this MIC cyanide issue might well prove to have been heavily influenced by semantics and the confusion between cyanide and methyl isocyanate. This sort of confusion would be quite understandable, even among medical people, in the early hours of a disaster caused by a hitherto toxicologically unknown compound. Whatever the reasons for the cyanide theory, the MIC disaster underlines, besides many other important issues already discussed in the *Journal* (1985;42:477-8), the need for accurate documentation and rapid scientific publication of clinical and laboratory findings after chemical accidents. This could be facilitated by effective international cooperation and close collaboration with experimentalists, as

References

- 1 Nemery B, Dinsdale D, Sparrow S. Methyl isocyanate: thiosulfate does not protect. *Lancet* 1985;ii:1245-6.
- 2 "Bhopal: industrial genocide?" Hong Kong: Arena Press, Asian Regional Exchange for New Alternatives, 1985.
- 3 Hamlyn M. Bhopal: was cyanide the culprit? *The Times* 1985 17 January.
- 4 Agarwal A. The cloud over Bhopal. *New Scientist* 1985; Nov 28: 38-41.
- 5 Anonymous. New Bhopal dispute: cyanide poisoning of victims claimed. *Chemical and Engineering News* 1985;63:6.
- 6 Aldridge WN, Connors TA. Chemical accidents and toxicology. *Hum Toxicol* 1985;4:477-9.
- 7 Lauwerys R, Louis-Ferdinand R. Human toxicology: the need for a closer collaboration between clinical and experimental toxicologists. *Hum Toxicol* 1984;3:61-2.

Chrysotile asbestos exposure and mesothelioma

SIR,—The recent paper by Gardner and colleagues (1986;43:726-32) highlights the continuing controversy regarding the carcinogenic potential of chrysotile asbestos. As the authors point out, although available data suggest that amphibole fibres are more potent for producing mesothelioma than chrysotile, this relation is not well defined for lung cancer.¹ The recent observation of a statistically significant excess mortality due to lung cancer among textile workers exposed exclusively to chrysotile is also of interest, suggesting that differences in airborne fibre characteristics may partially account for differences in mortality between chrysotile exposed occupational groups.²

This latter point may be important in that exposure to chrysotile fibres that have been physically "altered" due to industrial processing or through consumer use may prove hazardous. For example, to my knowledge no systematic investigation of brake repair workers, exposed solely to chrysotile, has been conducted, although some evidence exists which suggests that this occupational group may be at risk for developing mesothelioma.³

As part of an epidemiological study of diffuse pleural mesothelioma being conducted at the Canadian Tumor Reference Center, I have recently completed a preliminary review of 37 cases of this tumour associated with occupational asbestos exposure. (All cases were confirmed at necropsy.) Of these, three (all male) had job titles of "brake mechanic (auto)," "elevator

mechanic," and "railroad brake banding." These cases ranged in age from 56 to 65 with exposure histories of one month, 30 years, and 36 years. Two cases had sarcomatous tumours whereas the third had a tumour of the biphasic type. At necropsy all were found to have distant metastases.

Interestingly, at necropsy, tissue samples from the pleura of the subject identified as an auto mechanic were examined by an expert pathologist (by electron microscopy) to assess the mineral fibre content. Three pleural specimens were analysed, producing a range in fibre concentration of 51 to 266×10^6 fibres per gram of wet tissue, 99% of these fibres being chrysotile (1% amosite). These data are also interesting in that asbestos dust exposure during brake repair has been considered low by some investigators.⁴ Clearly, other occupational and non-occupational groups exposed to chrysotile asbestos should be investigated to define more accurately the risk of developing mesothelioma.

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References

- 1 Huncharek M. The biomedical and epidemiological characteristics of asbestos related diseases: a review. *Yale J Biol Med* 1986;59:435-51.
- 2 Dement JM, Harris RL, Symons MJ, Shy CM. Exposure and mortality among chrysotile asbestos workers. Part II: mortality. *Am J Ind Med* 1983;4:421-33.
- 3 Langer AM, McCaughey WT. Mesothelioma in a brake repair worker. *Lancet* 1982;ii:1101-3.
- 4 Rodelsperger K, John H, Bruckel B, Manke J, Paur R, Weitowitz HJ. Asbestos dust exposure during brake repair. *Am J Ind Med* 1986;10:63-72.

Notices

Principles of Epidemiology, Washington, 28-30 October 1987

The New England Epidemiology Institute will present a three day course on "Principles of Epidemiology." Topics will include modern concepts in epidemiology and their applications to the study of aetiology, natural history of disease, and strategies in preventive medicine and public health. No previous study of epidemiology or biostatistics is required. Registrants may receive Continuing Medical Education credits (AMA Category 1) through the Postgraduate Medical Institute or Certification Maintenance credits from the American Board of Industrial Hygiene, or both. For more information, contact: The New England Epidemiology Institute, Dept SC-7, PO Box 57, Chestnut Hill, MA 02167, USA.

Seventh Annual Epidemiology Summer Program, Medford, MA, 26 July-14 August 1987

The New England Epidemiology Institute and Tufts University will sponsor a three week summer programme in epidemiology at Tufts' Medford, MA, campus. The programme includes both methodological and substantive courses intended for those seeking an introduction to modern epidemiological concepts as well as those desiring a review of recent developments in epidemiological thinking. Thirteen courses will be offered, including theory and practice of epidemiology (introductory and advanced levels), biostatistics, regression and categorical data methods, logistic regression and survival analysis, research management and microcomputers in epidemiology, clinical epidemiology, cancer epidemiology, nutritional epidemiology, environmental and occupational epidemiology, reproductive and perinatal epidemiology, and epidemiologic bases of public health policy and law. Registrants may receive graduate degree credit, Continuing Medical Education credit (AMA Category 1) through the Postgraduate Medical Institute or Certification Maintenance credits in industrial hygiene. For more information contact: The New England Epidemiology Institute, Dept SC-7, PO Box 57, Chestnut Hill, MA 02167, USA.

Stress Management Programmes at Work, Loughborough, 1-2 June 1987

This workshop is designed for middle and senior managers in medium to large organisations. Its practical nature makes it relevant for both training and personnel managers responsible for staff development and technical managers and professional health and safety advisers responsible for job and work station design and for health policy. For further details contact: Anne Hill, Centre for Extension Studies, Loughborough University of Technology, Loughborough, Leics LE11 3TU. Telephone: Loughborough (0509) 222158.

Industrial Audiometry, Manchester, 13-15 May 1987

The course offers training in audiometry for industrial medical staff, safety officers, and others concerned with hearing in industry. It covers both the theory and practice of audiometry, together with lectures on audiometric methods, the accuracy of data, interpretation of audiograms, assessment of audiograms, and legal aspects. Practical work will include the use of manual, self recording and computerised instruments. Further details and application forms from: Dr W Tempest, Kismet, Croyde Rd, St Annes, Lancs FY8 1EX.